

REPORT ON THE PHYSIOLOGICAL EFFECTS OF SUDDEN CHANGES
IN THE SPEED AND DIRECTION OF AIRPLANE FLIGHT

by

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INTRODUCTION

A body at rest upon the surface of the earth is subjected to a pull of the earth's gravity proportional to its relative weight. The speed of a freely falling body adds to this normal pull by the development of an increased inertia. Depending upon the relative weight and the speed of falling, a body may attain a force many times that of normal gravity. If the straight course of a falling body is altered, the exaggerated tendency to continue downward will persist as an "acceleration" along the line of the direction of gravity. The strength of the force which develops will depend upon the inertia at the time of the change in direction and the abruptness with which the change occurs, and will be in multiples of relative weight -- the normal force of gravity known as gravity units, "g." Such an "acceleration" will be balanced by a "deceleration" in the speed of descent. Accelerations and decelerations are expressed in multiples of gravity, "X g."

When the earth's gravity has been overcome by the flight of an airplane and is replaced by the inertia of its progress through the air, change in direction of flight will result in a centrifugal force which is an acceleration, independent of the direction and force of the earth's gravity. This acceleration will be along the initial line of flight, that is, along the radii of the curve of change in direction. Such an acceleration will

be produced by any manoeuvre involving change in direction of flight. The magnitude of the acceleration will depend upon the speed at the time of change and the abruptness with which it is made.

For structural and aerodynamic reasons, it is highly desirable if not essential that accelerations of this nature always pass through the center of gravity along a "normal" line, i.e., along the line of normal gravity in the normal level flying position or the position at rest on the ground. In the proper execution of a horizontal turn, the plane is banked in such a way that the acceleration developed by the change of direction passes through the normal plane -- the plane at right angles to the line of flight and the plane of the wings. In recovery from a dive, the nose of the plane is pulled "up" (in respect to the level flying position) so that the acceleration passes through the normal plane. The acceleration is spoken of as "plus" if it is down in respect to the level flying position, and "minus" if it is up in respect to that position.

In the conventional design of aircraft, this normal line is parallel to the long axis of the pilot's body. With increased speed and structural strength, accelerations of many times gravity are encountered. Accelerations of 10 g. and more are not unusual in modern planes. This means that the pilot will be forced into his seat by a force equal to ten or more times his weight, and all his tissues and organs will be subjected to an equal acceleration. Physiological

changes can well be anticipated.

THE PHYSIOLOGICAL PROBLEM

The only uniform symptom attending sudden high accelerations is the one popularly known as "going black," "blacking out," etc. Coming on abruptly toward the end of a "pull out" and lasting for a variable period after the resumption of normal flight, there occurs a sensation of loss of vision. This is not so much a reduction of visual acuity because of blurring or fogging as a sensation of dimness due to reduced illumination. In the experience of the observer in this research, it is not unlike the progressive narrowing of the fields of vision experienced toward the end of a rebreather run. It comes on very abruptly and usually disappears quite as quickly.

The symptom is not constant in all individuals nor under all circumstances. It is exaggerated or diminished by certain conditions. There is considerable individual variation in susceptibility, some pilots complaining of pronounced "blacking" while others profess to never feeling it. It is more pronounced under reduced general fitness by fatigue, loss of sleep, etc. Familiarity reduces the symptom. If dives are repeated on successive days the symptom becomes less noticeable. It becomes more pronounced, however, if dives follow in quick succession. A series of frequent dives of moderate acceleration without symptoms may be followed by a very bad experience

attending a dive of even less acceleration.

The symptom is considerably modified by the configuration of the curve of acceleration. The same acceleration lasting over several seconds is much more apt to produce the experience than if acting for a short period. It has been reported that "a pilot may not lose vision in taking 10 g. for a short period (1 second), whereas, he may go black in taking 5 g. for a longer period."

Consciously increasing the abdominal and thoracic pressure will ameliorate the symptom. A high-pitched yell in which the intra-abdominal and intrathoracic pressures are raised by tensing the muscles of the abdomen, chest, and diaphragm against a partially closed glottis has been found very effective in preventing "going black."

Symptoms of abnormal "minus" accelerations have been reported. The opposite of "blacking out" has been described as "redding out" by the British. Subconjunctival hemorrhages have been seen after violent manoeuvres involving high "minus" accelerations. Capillary hemorrhages under supporting straps over the shoulders are seen after strenuous inverted flight.

Reports would indicate that the symptom is not attended by any other noticeable manifestation. There is no reduction in hearing, no loss of consciousness, no impairment of muscular strength or coordination. Pilots

who have experienced both insist that it is not at all like fainting or being knocked unconscious by a blow. It is very likely that other symptoms would be found if observation could be sufficiently accurate and searching. It has been the experience during this research that data which were entirely dependent upon the observer's judgment were not always reliable. There seems to be some reduction in attention and judgment in addition to dimness of vision.

Possible explanation of symptoms. The accelerations encountered during these observations operate equally on all parts of the body. Ligamentous and bony support of the solid tissues are adequate to maintain their normal position. There is absolutely no confirmation of reports that suspensions of viscera have failed, although it is not inconceivable that a turgid liver or kidney or a full hollow viscus might strain its suspension to the point of rupture.

Fluid components, being free to move, can be changed in distribution and relative pressure. The elasticity of the retaining walls will permit an accumulation of fluids in the areas on the outside of the turn at the expense of those on the inside. This applies particularly to the cerebrospinal fluid and the blood.

It is possible that the sudden application of an acceleration to the cerebrospinal fluid may result in disturbed normal support of the brain and an increase in

the pressure of the brain against the floor of the cranium. It is further possible that the medulla oblongata might be forced further than normal into the foramen magnum.

Under the influence of positive accelerations the blood will tend to accumulate in the lower parts of the body at the expense of the head.

The venous column of blood is supported by the valves in the pelvic region, the vasomotor tone, and the negative pressure in the chest. Positive accelerations applied to this column are capable of throwing all the venous blood into the lower part of the abdomen by engorgement of the systemic and portal systems. The venous pressure in the cranium will be correspondingly reduced and there will be reduced filling of the auricles. The increase in venous pressure in the lower part of the abdomen will impede the return flow from the legs and will cause a rise in venous pressure.

Conditions in the arterial system will be altered by the acceleration, but due to the higher pressure and greater elasticity of the arteries, the effect will be relatively less than that in the veins. Arterial pressures will be more influenced by secondary effects. The reduced filling of the auricles will reduce arterial pressure by reducing cardiac output. The carotid sinus will be activated by the change in carotid blood pressure,

causing reflex vasomotor changes. Reduced pressure in the sinus will cause vasoconstriction and tachycardia.

The significant results of the circulatory effects will be registered principally in the intracranial circulation. Both the venous and the arterial pressure in the cranium will be reduced by the simple hydrostatic effect and the reduced cardiac output. These changes will result in an anoxemia of the brain.

The increased venous pressure in the abdomen and extremities will cause an increased passage of fluid into the tissue spaces which will reduce blood volume. This will become significant if accelerations are applied at frequent intervals, and probably explains the increased effect of a series of dives.

"Minus" accelerations will, in general, reverse the process. Venous blood will surge upward, increasing intracranial pressure and increasing the supply to the auricles. Arterial pressure in the head will be increased by the hydrostatic effect and, secondarily, by increased cardiac output. Cerebrospinal fluid will tend to increase in the cranium at the expense of the spinal column. Intracranial pressure will be raised and the blood flow increased. The carotid sinus will initiate compensatory adjustments.

In the presence of reduced intracranial pressure and blood flow, the symptom of "going black" can be explained on the basis of either a retinal ischemia or intracranial

anoxemia.

PLAN OF INVESTIGATION

On the basis of the foregoing discussion, it was postulated that the arterial pressure should show changes resulting from the application of accelerations along the long axis of the body. If plus accelerations centrifuged blood into the lower parts of the body, this would be shown by changes in the arterial pressure. These changes would result directly from the hydrostatic influences and indirectly from adaptive changes as a result of normal circulatory control by the nervous system. It was decided to determine the arterial pressure under varying accelerations.

In the absence of practicable apparatus for determining rapid changes in blood pressure in man, it was decided that direct arterial pressure determinations be made on animals. The dog was selected because of size and because of our rather detailed knowledge of his circulatory reactions.

Accelerations might have been induced by elaborate and costly apparatus which could not, at best, simulate those induced in an airplane. To accomplish the desired accelerations it was absolutely essential that there be complete independence of the earth's gravity. Experience has shown that this is practically impossible when rigid contact with the ground is maintained. The damping effect of progress through highly compressible air could

not have been simulated except at prohibitive cost. Information was desired on physiological changes which occur during a few seconds of flight, and this could not be obtained except under actual flying conditions.

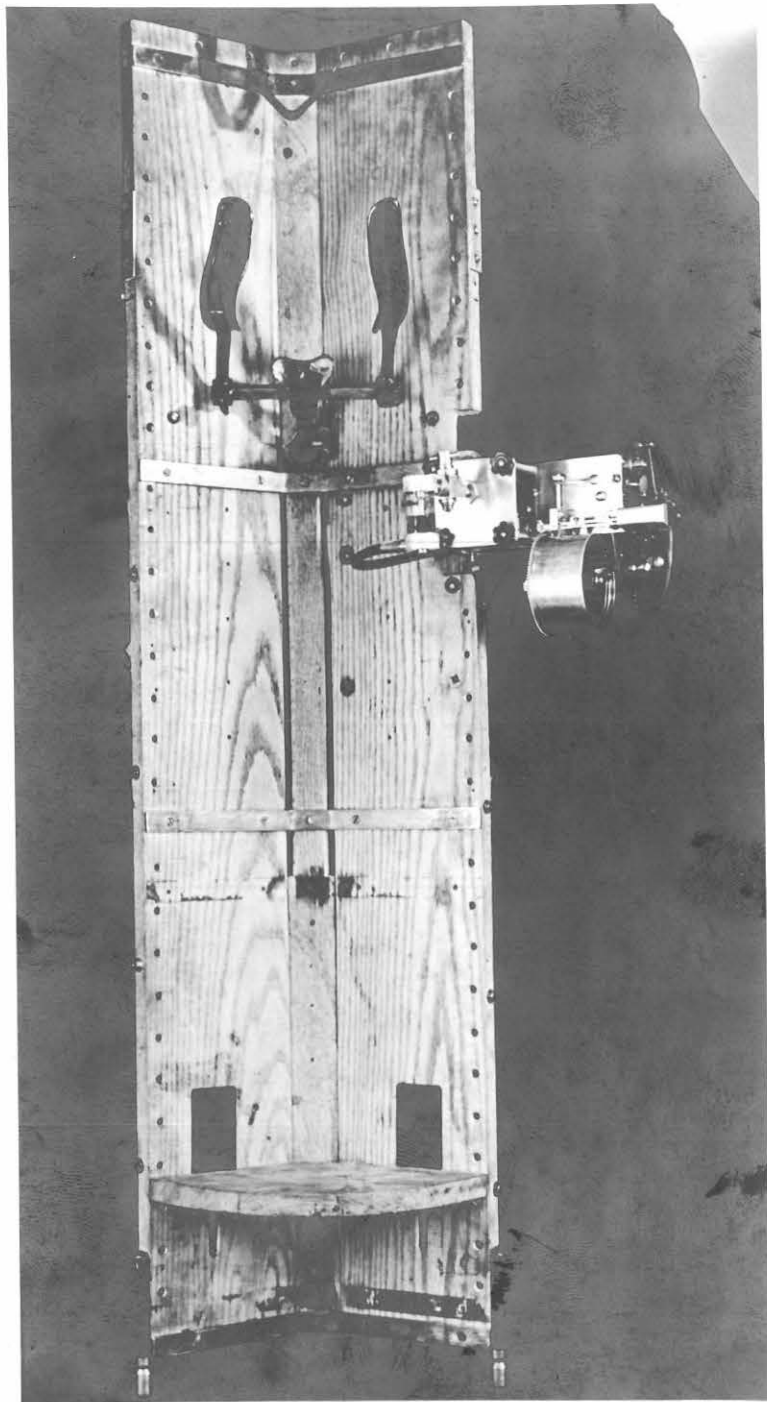


Fig. 1. The board with recorder attached.

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THE RECORDING APPARATUS AND THE ARRANGEMENTS IN THE AIRPLANE

A trough-shaped board (Fig. 1) was constructed of strong wood, appropriately braced by steel straps. It was equipped with a stationary rigid head-piece and an adjustable seat. The head-piece was made of steel, and consisted of a plate designed to fit under the dog's occipital protuberance and of adjustable pieces to fit under the mandibles. This gave rigid support to the head, did not interfere with respiration nor circulation, and kept the dog's head in a normal position. The seat was adjustable to varying lengths and gave support to the spinal column through the pelvis, which was kept in position by light straps. A canvas jacket across the chest was buckled to the back of the board and gave support in this direction without interfering with respiration. An elastic and canvas support was buckled across the abdomen but exerted no pressure.

The board was fitted with two half-inch steel pins at the bottom, which were screw clamped into sockets bolted into the floor of the plane. Two slotted brackets slipped over a cross member clamped across the front of the after cockpit opening. With this arrangement it was possible to mount the assembly into the plane without undue effort or loss of time.

As a protection against cold, a flannel-lined leather

bag was constructed which completely covered the dog and board. This was provided with "zipper" fasteners along the front to permit adjustment of supports, intravenous medications, etc., without removing the covering.

Certain unusual circumstances presented themselves in the design of a recording apparatus. Compactness and conservation of size and weight were essential. Controls must be easily accessible. The recorder must be as automatic as possible because of the fallacy of personal observations. There must be provision for taking several records on a single flight.

Availability of space precluded the use of any of the conventional recording apparatus such as the familiar laboratory kymograph. The mechanism of translating the pressure in the artery to the writing element must be free from any possibility of being itself activated by the accelerations. This precluded the use of a fluid manometer or a spring manometer so placed that its tension would be materially affected by the direction and force of the accelerations. To minimize hysteresis there should be fluid continuity between the arterial column and the translation mechanism.

Fortunately, accelerations which could be of physiological significance were all in the vertical plane, so that if the moving parts of the recorder

operated in the horizontal plane there would be no acceleration artefacts.

The first recorder embodied the principle of translating the movement of a rubber tambour into movement of a writing point recording on a small smoked drum moved by clockwork. The tambour was directly connected with a T-cannula inserted in an artery so as to preserve fluid continuity throughout. It was covered with rubber and so mounted on a base plate that the rubber diaphragm lay in the horizontal plane. The depth of the tambour never exceeded one inch at its greatest distension.

As the writing point moved across the smoked paper revolving under it, a direct graph of variations in the pressure in the tambour was obtained. By careful calibration of the movement of the diaphragm under known successive increments of pressure, it was possible to determine the actual pressure at any point on the graph.

A simple manually-operated signal marker was installed which also provided a base line on the graph.

Repeated tests of the recorder under different accelerations showed it was free from artefacts, and that any variation in the graph was the result of variation in the arterial pressure of the animal and not due to movement of the recorder itself. A number of very satisfactory records were made with this recorder. Qualitative trends in the arterial pressure were clearly

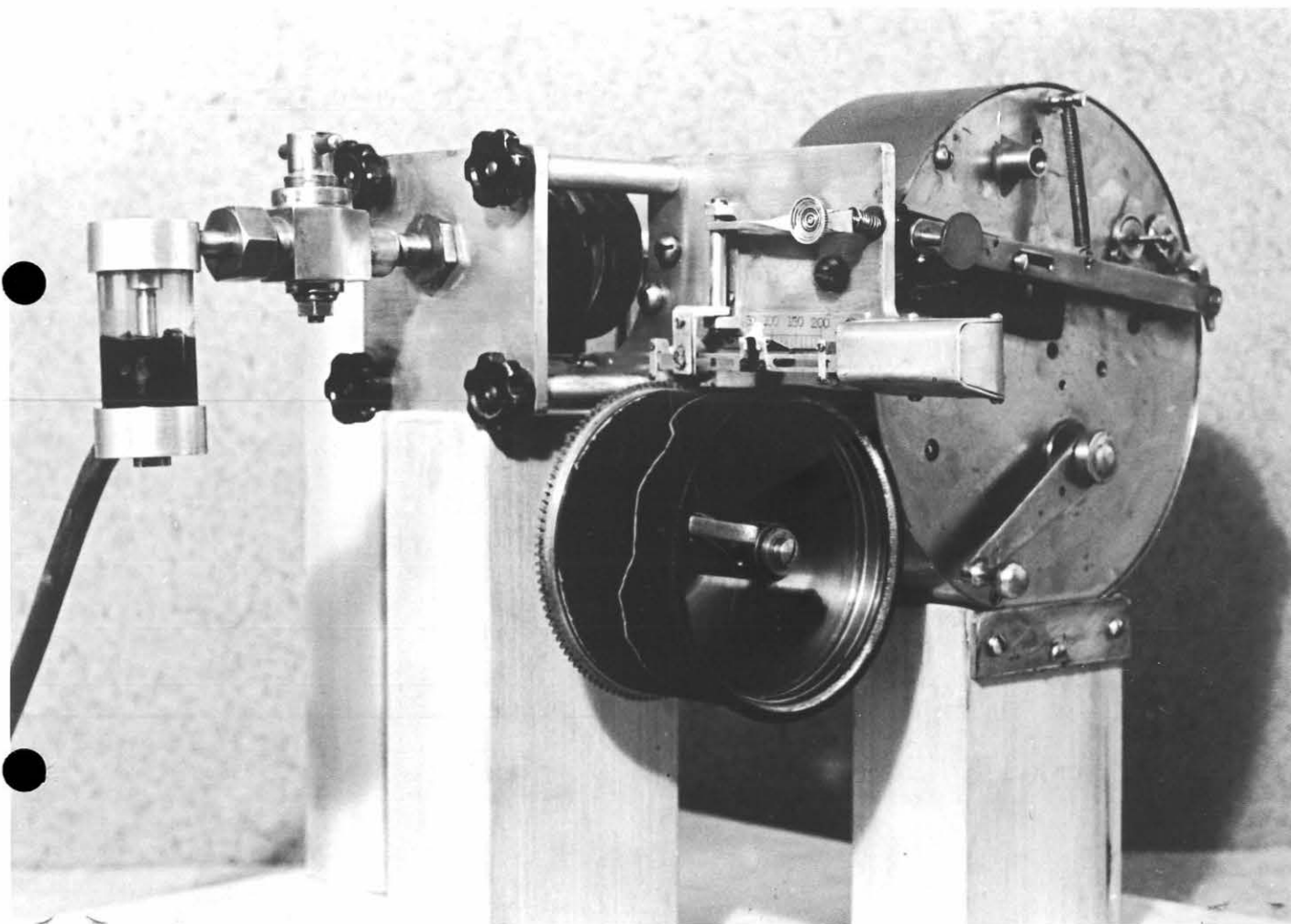


Fig. 2. A, separation chamber; B, valve; C, tambour; D, drum; E, event marker; F, writing point; G, writing-point carrier; H, starting lever; I, kymograph winding lever.

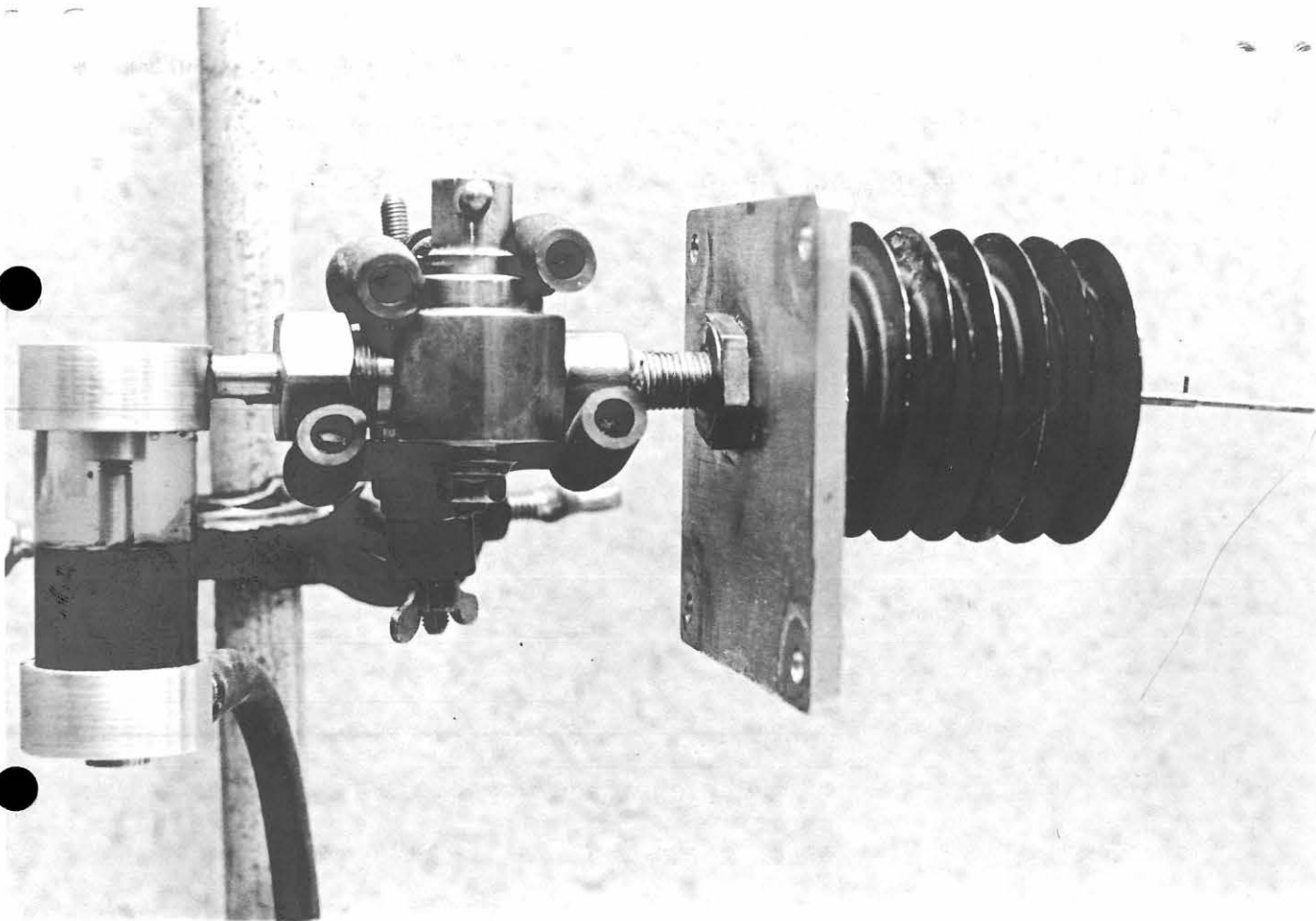


Fig. 3. Showing the separation chamber, valve, and bellows type tambour. The horizontal guide pin attached to the free end of the tambour carried the vertical pin which engages the slot in the short arm of the bell crank of the transmission mechanism shown in Fig. 4.

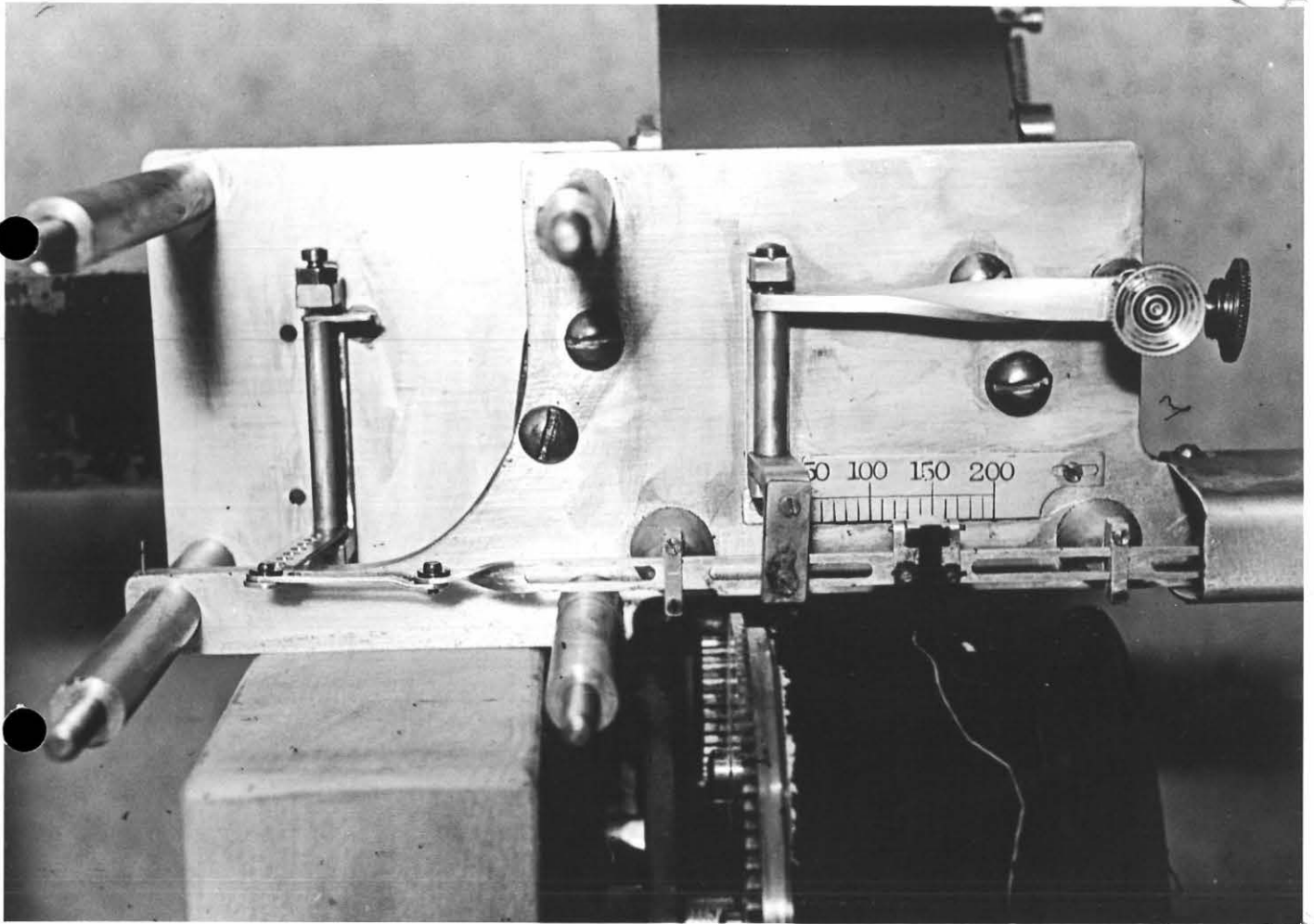


Fig. 4. Showing the transmission mechanism which serves to amplify and change the direction of the movement of the tambour to the writing point.

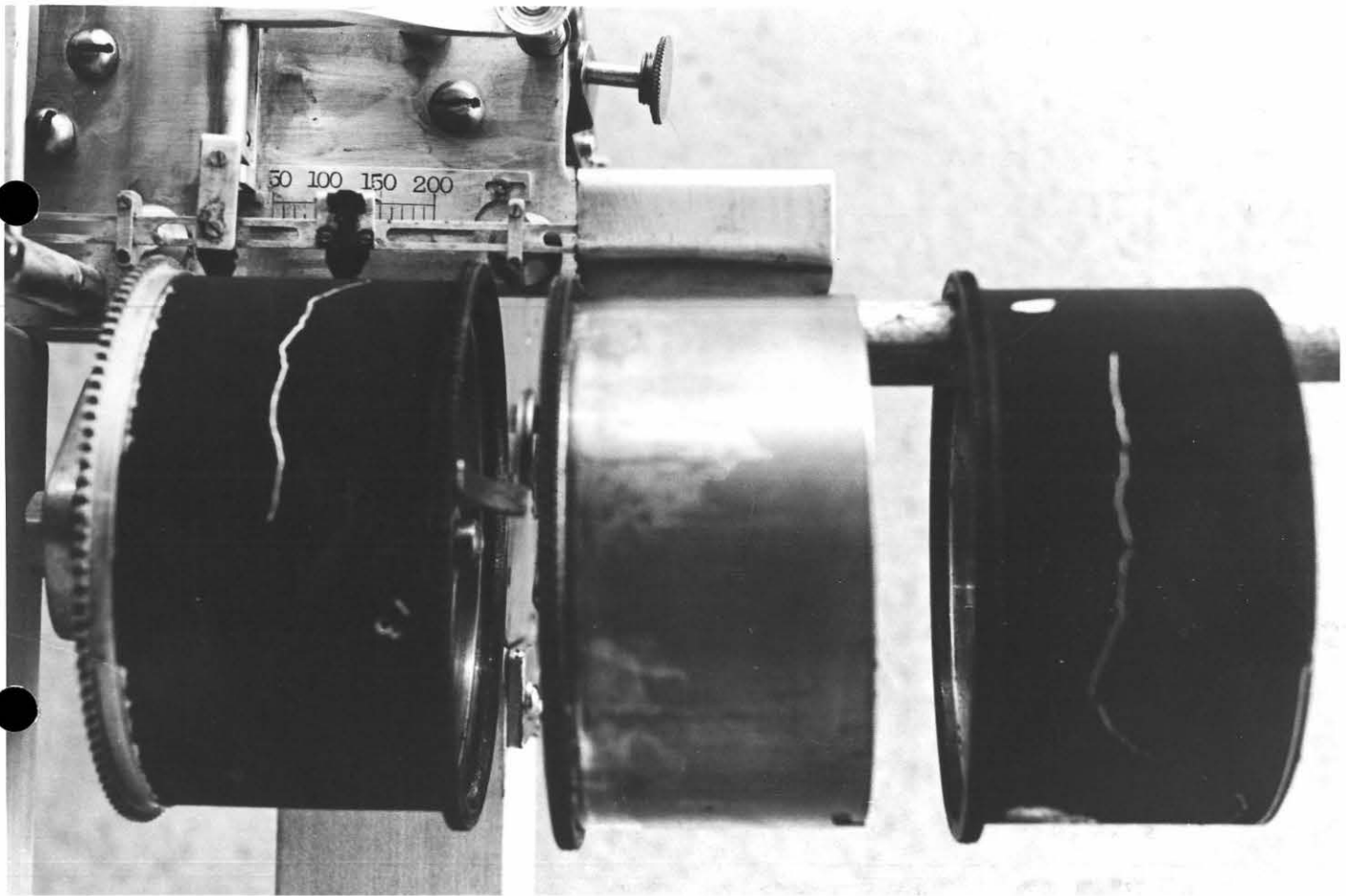


Fig. 5. Showing the drum and two additional sleeves, one with and one without smoked paper.

shown. An objection to the use of rubber diaphragms became apparent when it was decided to ascertain quantitative differences more accurately. Variations in the elasticity of different grades of rubber, fatigue and deterioration, hysteresis, and the necessity of careful calibration before each experiment prompted the development of a recorder in which these objections were removed.

The second recorder is shown in the accompanying photographs. After much experimentation a bellows-type tambour was made from thin spring brass. Thin disks of brass were spun to the desired shape and size and soldered into lozenges. A group of six lozenges comprised the tambour.

The details are shown in Figures 2 to 5. The entire assembly is shown in Figure 2, which indicates the relationship of the tambour to the writing point and to the drum on the kymograph. The details of the tambour are shown in Figure 3. A valve was incorporated to permit cutting out the recorder when not in use. To prevent fouling of the tambour by blood from the animal, there was inserted a chamber which separated the oil with which the tambour was filled from the connection to the animal. The openings of this chamber were so placed that separation was maintained when the apparatus was either horizontal or vertical.

A guide pin soldered on the free end of the tambour

moved through the guide-pin bearing in the base plate. This restricted movement to the longitudinal axis of the tambour. On this guide pin was mounted a short vertical pin which engaged a slot in the short arm of the bell crank of the transmission mechanism, shown in Figure 4. This crank served to amplify the movement of the tambour six times and change the direction of movement to conform to the relationship between the writing point and the drum. This figure also shows the details of the event marker and the gearing of the drum to the kymograph. In Figure 5, the method of mounting the sleeves on the drum is shown and two additional sleeves, one with and one without smoked paper (inadvertently reversed in this photograph). All moving parts of this recorder operated in the horizontal plane, i.e., at right angles to the direction of the accelerations encountered. This precluded records which might result from movement of the recorder itself.

Preliminary calibration established the constancy of the tambour under variations in pressure far in excess of those anticipated, and it was found justifiable to incorporate a scale for the anticipated range in pressure, 50 to 200 mm. Hg. This made it possible to follow variations in recorder pressure in terms of actual arterial blood pressure. It also eliminated one complete step in each experiment, viz., a control record in which the pressure indicated on the graph was checked against



Fig. 6. Dog and recorder mounted on board ready for mounting in plane.



Fig. 7. Dog and board mounted in the plane.

a mercury manometer reading.

This recorder has been found highly satisfactory and has produced records which not only indicate the nature of changes in arterial pressure but their numerical values as well. Great credit is due to the machinist of the Harvard Medical School, Mr. F. J. Christensen, for his skill and ingenuity in making a physiological instrument of entirely novel character which has behaved perfectly under the most trying conditions of experiment.

The recorder was supported on an arm which could be clamped into a slotted fitting on the back of the dog board. The dog and recorder assembled on the board and the whole mounted in the plane are shown in Figures 6 and 7. The unit is small and compact, adapts itself to operation in an airplane, is self-contained and automatic, easily accessible, and embodies all the principles enumerated in considering the requirements. The time error inherent in the spring-driven kymograph was reduced to a negligible minimum by winding the spring to the same tension before each run. By repeated practice the amount of winding necessary to insure this invariable tension after one revolution of the drum could easily be estimated. Within rather wide limits this tension gave constant speed. By starting the drum several seconds before the manoeuvre a satisfactory constancy of speed could be relied upon. Significant changes were found to embrace multiples rather than

fractions of seconds, and the time error was very safely within these limits.

The airplane used was the standard BM-2. Smoothness and ease of control makes this model particularly well adapted. It appears to be singularly independent of small extraneous influences such as bumps, grooves very well, and it is possible for a pilot with a little practice to produce at will practically any desired acceleration within the limits of the plane. These features were particularly desirable in those experiments in which the magnitude and the shape of the acceleration curve were important variables. The absence of V-G cards does not significantly reduce the assurance that accelerations produced were as desired and reported.

The N.A.C.A. recording Accelerometer was used to determine the magnitude of accelerations. Inasmuch as practically all experiments were more concerned with relative than actual accelerations, any errors inherent in this recorder do not in any way vitiate the findings. Recorded accelerations were taken from the records obtained on this instrument.

Choice of anesthetic was dependent upon ease of administration, constancy, lasting qualities, and absence of interference with the maintenance of normal vasomotor control in the erect posture. Preliminary experiments in the laboratory in which dogs were kept in the erect posture for long periods and their position quickly

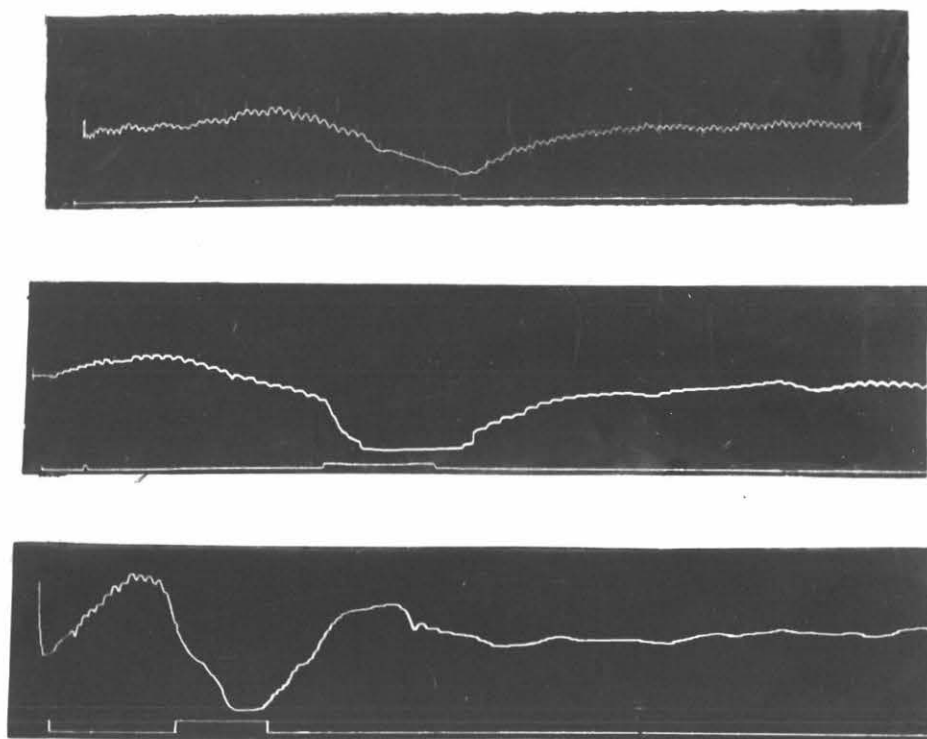


Fig. 8. Three typical graphs.

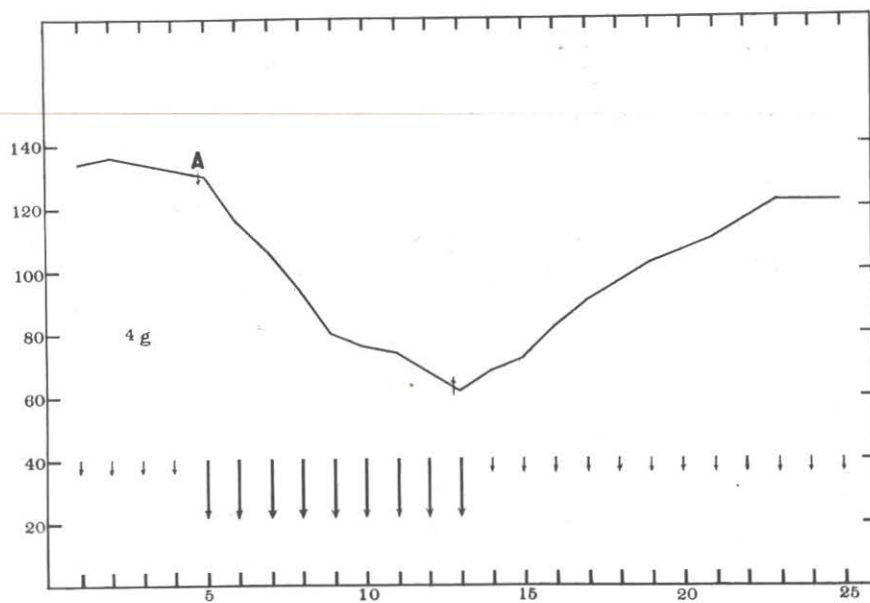


Fig. 9. The horizontal turn.

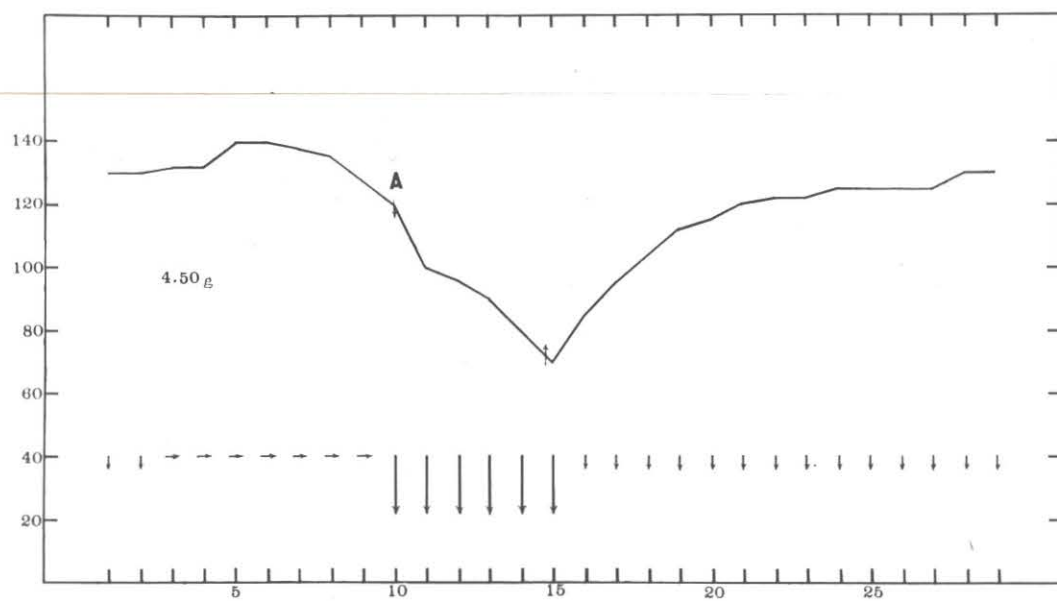


Fig. 10. The dive.

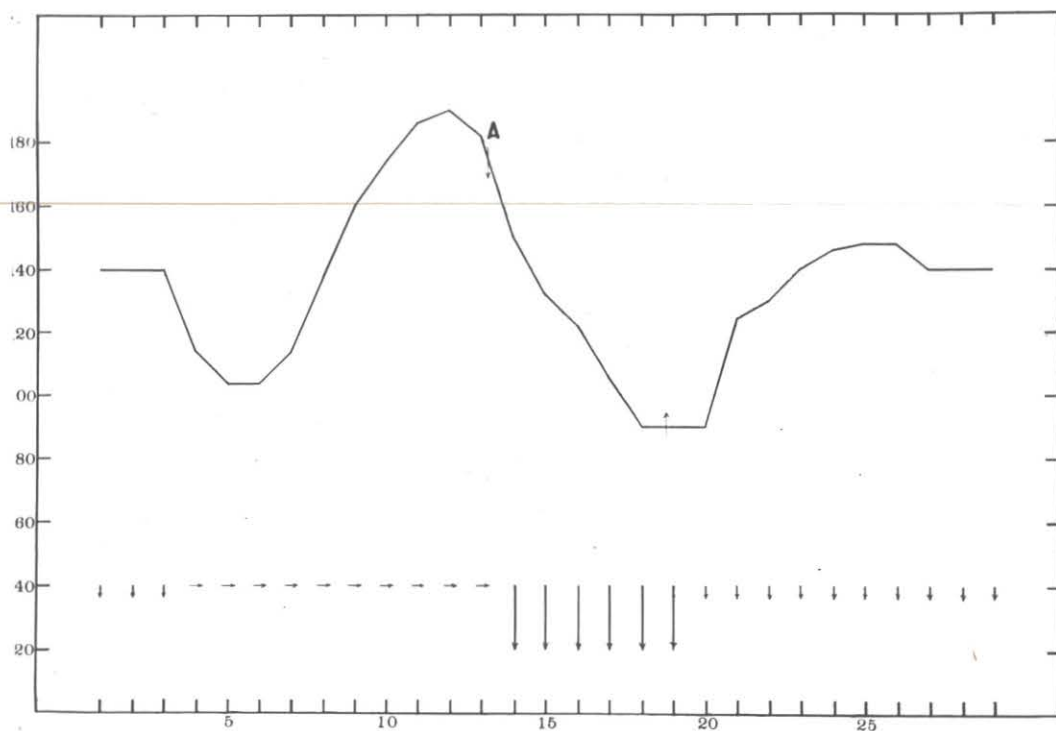


Fig. 11. A typical effect during the dive.

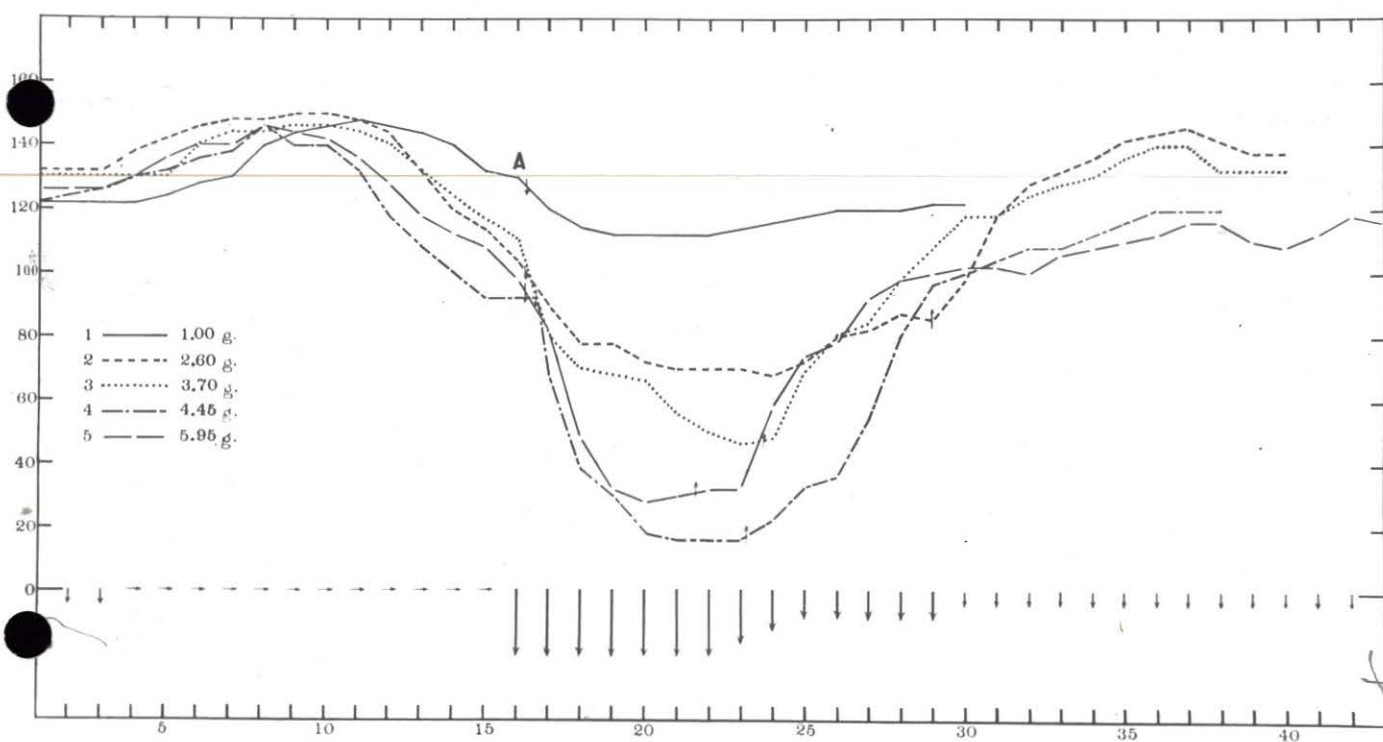


Fig. 12. Successive dives of increasing acceleration.

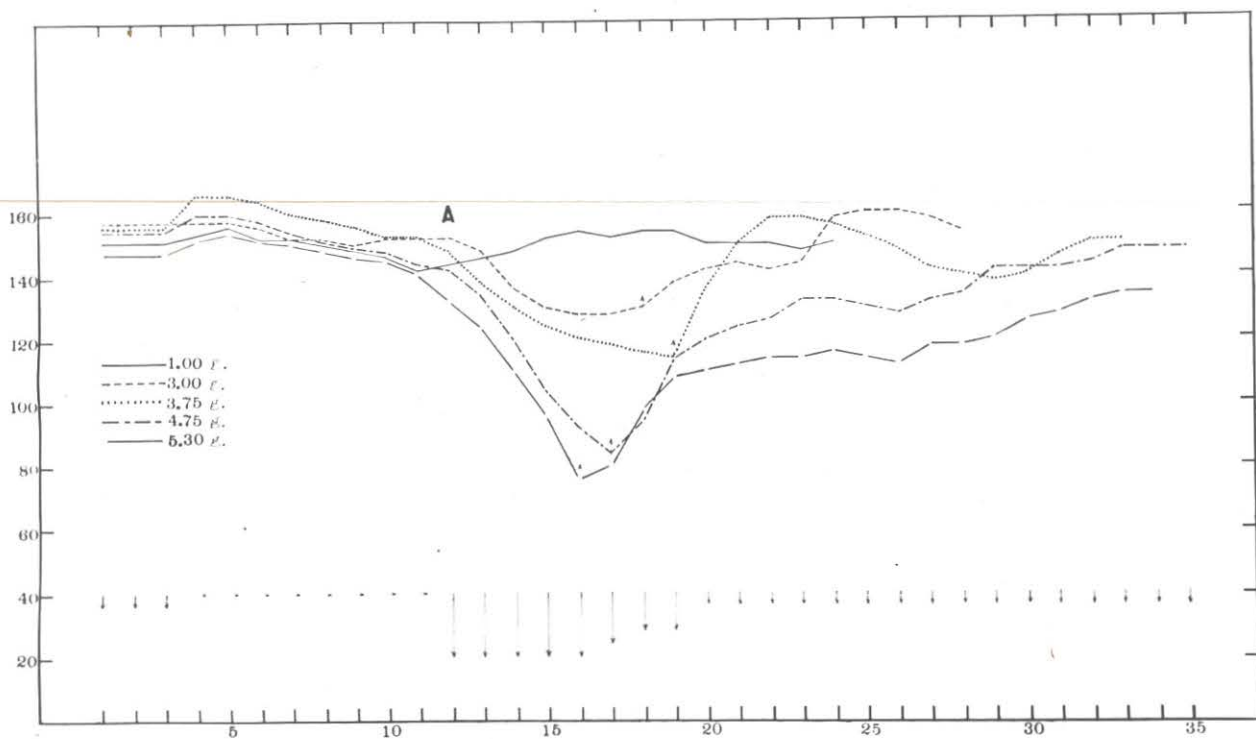


Fig. 13. Successive dives of increasing acceleration.

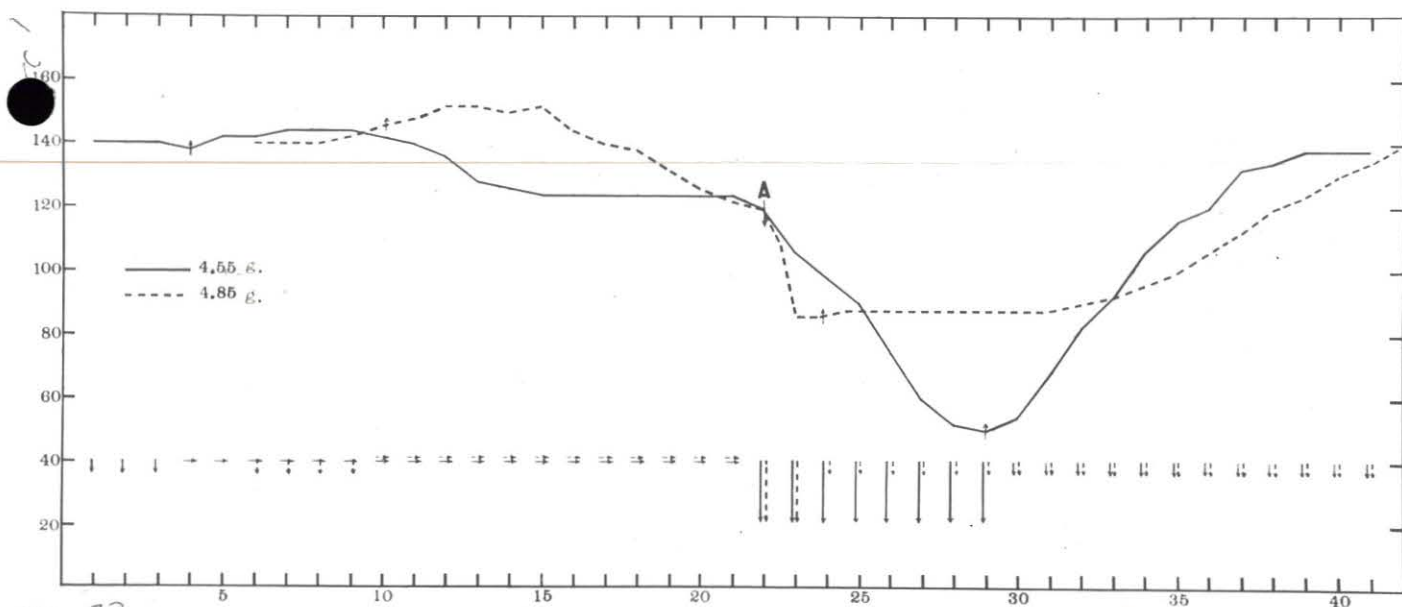


Fig. 14. Dives of approximately the same acceleration but of short and long duration.

changed from horizontal to vertical established the fact that the anesthetic chosen met these requirements. The intraperitoneal injection of an aqueous solution of Nembutal (Sodium-ethyl (1-methyl-butyl)barbiturate), in doses of 40 mg. per kilo of body weight, gave satisfactory anesthesia for several hours and did not materially interfere with vasomotor control. Compensatory vasomotor adjustments to very sudden changes in position were uniformly unimpaired. Compensation following the application of high accelerations was equally satisfactory. Usually one dose sufficed. Occasionally an additional small dose was found desirable later in the course of an experiment.

Intravenous heparin in doses of 1 mg. per 5 cc. of blood was used as an anticoagulant. Clotting resulted in spoiled records in only two experiments.

Preparation for aerial experiments consisted of two stages, one at the laboratory and one at the flying field. The dog was anesthetized and fastened to the board, a cannula was inserted in the femoral vein and the left carotid artery was isolated at the laboratory. At the field the dog was heparinized, the T-cannula inserted in the carotid artery, and the recorder attached and checked. With as little delay as possible the apparatus was mounted in the plane and the flight begun.

THE EXPERIMENTS

The desired manoeuvres were begun as soon as sufficient altitude had been gained and were repeated as quickly as operation of the recorder permitted. This assured maximum use of the anticoagulant and short intervals between runs.

Unexpected variations in the ability of the dogs to withstand the experience were encountered. In spite of the care in selecting only healthy, fairly large animals, some reacted poorly to moderate accelerations. One apparently healthy animal whose circulation was in splendid condition on taking off failed to survive two dives and was dead on landing. Another survived fifteen dives of average magnitude with the help of intravenous saline and ephedrin after seven of them. Still another withstood seventeen dives without intermediate medication, and was still in good condition when both the pilot and observer had had enough for the day. All animals were sacrificed upon the termination of the experiment. Arterial pressures were investigated under the following conditions and will be discussed in some detail under individual headings: (a) the horizontal turn, (b) the dive, (c) successive dives of increasing acceleration, (d) dives of approximately the same acceleration but of short and long duration, (e) the inverted turn, (f) the effect of atropine and ephedrin, (g) the effect of an abdominal binder, and (h) the effect of varying pressures

against the abdomen.

A uniform type of plotted graph was constructed for all experiments, in which the ordinates represent time in seconds and the abscissae arterial pressure in millimeters of mercury. Arrows indicate the direction and amplitude of accelerations at each second. Short vertical arrows indicate normal gravity in the normal direction. Horizontal arrows indicate that normal gravity was released, as during a dive, and is considered physiologically zero. Long heavy arrows indicate the periods during which accelerations were more than normal (1 g.), "plus" when pointing downward and "minus" when pointing upward. The letter "A" indicates the second in which increased accelerations developed. Arrows across the line of arterial pressure indicate the exact point at which increased accelerations began (down) and ended (up).

(a). The horizontal turn. Figure 9 shows a characteristic graph of the arterial pressure during a turn in which the acceleration reached a maximum of 4 g., and remained fairly uniform during the turn. The dog was mounted in the upright position, i.e., with the long axis of his body in a plane parallel to the long axis of the pilot's body.

The first four seconds indicate the time before the turn was begun. At the fifth second the plane was flown into a sharp ninety-degree turn and was held in the turn for nine seconds, including the thirteenth, at which time it was quickly rolled back to normal level flight. The

only gravitational, centrifugal change during this manoeuvre was the abrupt introduction of plus 4 g. at the fifth second and an abrupt return to normal at the thirteenth.

The pressure in the carotid artery dropped from 130 mm. Hg with the introduction of the increased acceleration and continued dropping until it reached 62 mm. Hg at the end of the turn, after which it returned gradually to 122 mm. Hg.

In our opinion, drop in pressure is certainly due, first, to the centrifugal, hydrostatic effect of the blood being thrown from the head into the lower parts of the body and, second, to reduced cardiac output because of reduced venous supply to the auricles. The return to normal is due to reflex vasoconstriction and tachycardia, in response to stimulation of the carotid sinus by the reduced pressure and in response to direct stimulation of medullary centers by anoxemia.

(b). The dive. Figure 10 shows the change in arterial pressure during a dive in which the acceleration reached 4.50 g. in the pull out.

At the third second the plane was allowed to fall into a zero lift dive. Normal acceleration became zero. The dive continued to the tenth second when the plane was pulled out of the dive by changing course through horizontal to ascending flight. At the fifteenth second the plane resumed normal level flight. Accelerations, as shown by the arrows, were: first and second seconds,

1 g. (normal); third to ninth second, inclusive, zero; tenth to fifteenth second, inclusive, 4.50 g.; after the fifteenth second, 1 g.

Upon the release of gravity the blood surged upward causing a rise in pressure from 130 to 140 mm.Hg. This stimulated the carotid sinus, and produced reflex vasodilation and slowing of heart rate as is evidenced by the beginning drop in pressure during the sixth and seventh seconds. The pressure continued to drop as a result of this compensatory vasodilation and had fallen to 120 mm. Hg at the time the pull out began.

In the presence of this vasodilation the application of the plus acceleration caused the pressure to drop and to continue to drop during the entire time of pull out, reaching 70 mm. Hg in the fifteenth second. Compensatory vasoconstriction resulted from this drop in pressure, but was ineffectual in overcoming the abnormal acceleration until that had been reduced by the resumption of level flight, when it caused the pressure to return to the normal level.

The course of events was the same as in the turn, with the additional factors of initial rise in pressure at the beginning of the dive and a vasodilation at the time of pull out.

Figure 11 shows the result in an animal with more active vasomotor control. In this case the dive was begun by wing-over, which produced a slight plus acceleration. The pressure dropped briskly as a result but

was quickly restored to the normal level by vasoconstriction. This compensatory vasoconstriction, added to the hydrostatic surge upward by the release of normal gravity in the dive, caused the pressure to rise to 190 mm. Hg. Although the pressure during the period of high acceleration dropped to only 90 mm. Hg, this is a drop of practically 100 mm. Hg from that at the time acceleration began. In returning to normal upon the resumption of level flight, the pressure rose to slightly above normal for a short period.

(c). Successive dives of increasing acceleration.

Figure 12 shows the effect of increasing accelerations. The first curve was made on the ground. Having been kept in the vertical position until the arterial pressure had stabilized, the dog was quickly lowered to a horizontal plane at the fourth second, maintained in this position until the sixteenth second, and then as quickly returned to vertical. Gravity effects on his long axis were: 1 g. until the fourth second, zero g. until the sixteenth, and then 1 g. for the remainder of the record.

Succeeding curves were obtained in the air in the sequence noted. All were dives with recovery by pull out. Each consisted in a dive from the third or fourth second to the sixteenth, followed by pull out in which successive accelerations were of increasing magnitude. The values for respective accelerations were taken from the N.A.C.A. recording accelerometer. The average

interval between dives was two minutes.

There was considerable variation in the time of pull out. Number 2, 2.60 g., was shallow and lasted thirteen seconds. Number 3, 3.70 g., lasted eight seconds. Number 4, 4.45 g. lasted five and one-half seconds. The time of pull out in number 5 is indicated as five seconds but, being dependent upon a subjective record by the event marker, cannot be relied upon. The observer went quite "black" and cannot vouch for the accuracy of the observation. From the appearance of the curve it is much more likely that the peak of acceleration lasted three seconds or less. This will be more apparent after studying the effect of short and long exposure to acceleration in subhead (d).

All curves show the typical configuration described for the simple dive. As all gravity is released during the dive the arterial pressure rises, to drop again as a result of the vasodilation caused by stimulation of the carotid sinus. Application of high acceleration causes a drop in pressure which varies with the magnitude and shape of the curve of acceleration, to be restored to normal by vasomotor control upon the cessation of the abnormal acceleration.

The relationship between the magnitude of the acceleration and the drop in pressure is shown in the following table:

<u>Acceleration</u>	<u>Drop in arterial pressure</u>
1.00 g.	36 mm..
2.60 g.	82 mm.
3.70 g.	100 mm.
4.45 g.	130 mm.

The progressive increase in the drop with increasing accelerations is consistent, varying only because of the manifest impossibility of accurately controlling the shape of the acceleration curve, which is a very significant variable.

These curves clearly show the increasing reluctance with which the arterial pressure returns to normal as increasing accelerations follow closely upon one another. The pressure returned to normal three seconds after 2.60 g., and ten seconds after 3.70 g. Fifteen seconds after 4.45 g. had been reduced to 1 g., the pressure had not returned to normal. Twenty-one or more seconds after 5.95 g., normal pressure had not been restored. This results not only from the relative severity of the individual dives but also from the fact that the pressure in the capillaries of the lower parts of the body is markedly increased, causing impaired return of extravascular fluid and stasis. There thus occurs a progressive increase of fluid in the tissue spaces and reduction of blood volume by fluid loss. This establishes a physiological basis for the fact that moderate accelerations are more apt to result in symptoms if they follow in rapid sequence.

The apparent inconsistency between the low points in numbers 4 and 5 is explained by the fact that the higher acceleration could not be maintained at its peak for as long as the lower. This places the two dives relatively in the class described in subhead (d).

Parenthetically, an interesting commentary upon circulatory and vasomotor efficiency is furnished in number 4, in which the arterial pressure of an anesthetized dog can be restored to a healthy level within a very few seconds after having been driven down to 16 mm. Hg.

The findings in this experiment were closely paralleled by the subjective experience of both the pilot and the observer. Number 5 on this chart was the seventeenth dive of an average acceleration of 4.20 g. within less than two and one-quarter hours. For three of the dives the observer was forced to stand erect in the plane. The severity of the "blacking out" together with the sense of fatigue almost to exhaustion made him ready to concede the dog the winner.

Figure 13 shows another experiment of the same nature and with essentially the same findings. The increasing reluctance to return to normal is even more striking than in Figure 12.

(d). Dives of approximately the same acceleration but of short and long duration. Figure 14 shows the change in arterial pressure in the same dog in two dives made within nine minutes of one another. In both, the

maximum acceleration was practically the same (4.55 g. and 4.85 g.). In one case the acceleration lasted for more than eight seconds and in the other for less than two seconds. In the first case the acceleration was gradually increased. In the second case it was very abruptly applied. In the first case the arterial pressure dropped gradually from 120 mm. Hg during the period of increased acceleration to as low as 50 mm. Hg. The return to normal was typical.

In the second case the pressure dropped abruptly to 86 mm. Hg during the peak of acceleration and then held at 88 mm. Hg for several seconds. Return to normal was leisurely.

The explanation for the difference lies in the fact that in the second case the secondary cause for drop in pressure did not develop, viz., reduced cardiac output because of decreased venous supply to the auricles. The acceleration lasted such a short time that pooling in the abdomen and lower extremities did not take place and the supply by the vena cavae was very little reduced. The drop in the second case resulted from purely hydrostatic, centrifugal forces. Apparently the drop was insufficient to initiate a prompt reflex vasoconstriction, which accounts for the fact that the pressure remained at a moderately low level for some time.

These two curves give graphic explanation for the observation that "a pilot may not lose vision in taking 10 g.

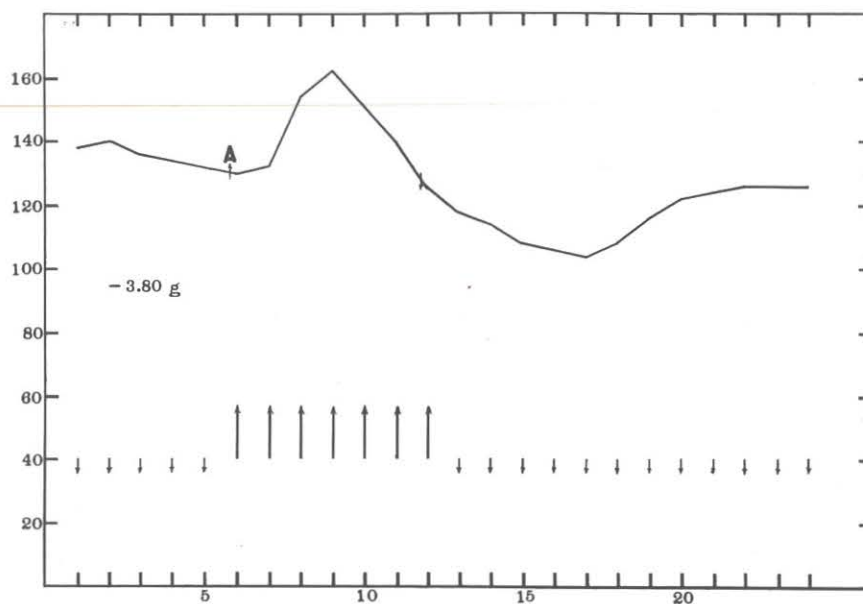


Fig. 15. The inverted turn.

for a short period (1 second), whereas, he may go black taking 5 g. for a longer period."

(e). The inverted turn. The plane used does not lend itself readily to a manoeuvre in which a minus acceleration of any magnitude could be relied upon as being unadulterated by other secondary accelerations. The likelihood of falling into a dive from an inverted turn was so great that if accelerations of significant magnitude were accomplished by this means they would most certainly be complicated by other effects. Accordingly, it was decided to make records with the dog mounted head downward in the plane. In this way the effect upon the animal in a normal turn which was easily accomplished would be that of an inverted turn of the same acceleration. Four records were made in this way, of which Figure 15 is typical. The turn was started in the sixth second and continued through the twelfth. After a slight delay the arterial pressure rose from 130 mm. Hg to 162 mm. Hg. Reflex vasodilation was prompt and the pressure had dropped to 126 mm. Hg before the turn was completed. The progress of vasomotor compensation after the turn is clearly shown.

(f). The effect of atropine and ephedrin. A dog weighing 28 kilos was given an intravenous injection of 98 mg. of ephedrin sulphate and 15 mg. of atropine sulphate. Except for raising the general level of pressure there was no material change in the shape of the arterial

curves before and after the injection. The response to the abnormal accelerations and compensatory vasomotor changes were quite the same as before the drugs were administered.

(g). The effect of an abdominal binder. On the supposition that restraint of the abdomen might prevent some of the pooling and thereby reduce the drop in pressure attending high accelerations, an experiment was conducted with this in mind. A semi-elastic webbed binder was constructed which could be snugly buckled around the abdomen. Dives and turns were made with this binder alternately loose and tight.

No material change in the shape and magnitude of the arterial pressure curves as a result of this restraint was noted. Apparently, simple restraint of normal abdominal pressure is not sufficient to prevent the drop in pressure.

(h). The effect of varying pressures against the abdomen. A rubber bag was fitted so that it could be supported over the abdomen in such a way that expansion would result in increasing the pressure against the abdomen. By inflating this bag, any desired pressure could be established. A few preliminary experiments were run to determine the effect of different pressure in controlling the drop in arterial pressure.

The results obtained give considerable promise that pressure against the abdomen of proper degree and applied

at the proper time may be found to result in materially reducing the effects of high accelerations.

This subject is worthy of further careful consideration and is proposed as the most promising of fruitful results in a search for methods of ameliorating the physiological changes found in this investigation.

The magnitude and certainty of the circulatory phenomena which have been described, coupled with the short time during which "going black" is experienced and the complete absence of symptoms following the experience, cause us to feel certain that no cerebral changes such as concussion, medullary pressure, or serious shifts in cerebrospinal fluid take part in the experience. The experiments reported leave no doubt that a transient cerebral anoxemia is the cause of the symptoms which have been described, and there is every reason to believe that with this knowledge as a basis, means can be supplied to counteract the sudden decrease in blood pressure which inevitably accompanies the dive and horizontal turn as now experienced in dive bombing or in the rapid horizontal turn.

SUMMARY

The intraperitoneal injection of Nembutal into dogs causes excellent anesthesia without impairment of circulation.

Dogs anesthetized with Nembutal were mounted in an airplane in a position exactly similar to that occupied

by the aviator and subjected to rapid horizontal turns and dives. Direct records of arterial pressure were made during these manpuevres.

A drop in arterial pressure occurs which is directly proportional to the severity and time of application of the abnormal forces. This causes a severe cerebral anoxemia which accounts for the symptom of "going black."